Effects of environmental variation during seed production on seed dormancy and germination

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Abstract

The environment during seed production has major impacts on the behaviour of progeny seeds. It can be shown that for annual plants temperature perception over the whole life history of the mother can affect the germination rate of progeny, and instances have been documented where these affects cross whole generations. Here we discuss the current state of knowledge of signal transduction pathways controlling environmental responses during seed production, focusing both on events which take place in the mother plant and those which occur directly as a result of environmental responses in the developing zygote. We show that seed production environment effects are complex, involving overlapping gene networks active independently in fruit, seed coat and zygotic tissues that can be deconstructed using careful physiology alongside molecular and genetic experiments.

Key Words: seed dormancy, seed development, seed germination, maternal, environment, temperature, flowering time, FLC, seed coat, tannin

Highlight: Environmental signals during seed production strongly affect seed dormancy at harvest and are exploited by plants to generate variation in dormancy levels among progeny. This review discusses the state of the art knowledge of the underlying biological mechanisms.

Introduction

It has been known for more than a century that the mother plant has a significant influence over seed traits, including seed size, dormancy and germination. In many species factors such as age of the mother plant, position of the seed in the fruit, inflorescence or canopy can affect seed properties, often accompanied by a dimorphism either of the seeds themselves or the fruits in which they arise (Roach and Wulff, 1987). In addition to the major effects of the genetics and developmental characters of the mother plant, the environment during seed

production also has a major influence on seed germinability (Fenner, 1991), and this will be the major focus of this review. These environmental effects can be strong and temperature is the most dominant signal across species, with as little as 1°C differences shown to have important consequences for seed dormancy in sensitive ranges (Springthorpe and Penfield, 2015). This extreme sensitivity stretches the capabilities of modern controlled environment technology in generating reproducible datasets experiment after experiment. This effect of temperature on seeds may be designed in part to transduce environmental noise into variation in dormancy among progeny (Springthorpe and Penfield, 2015) and therefore can be a confounding factor during commercial seed production, and severely restricts the global regions in which high quality seed can be produced for major seed markets.

Researchers typically refer to these environmental effects as the influence of the 'maternal environment' on seed properties, which has in the past been used to refer generally to the environmental conditions during seed production (Roach and Wulff, 1987). However, in the era of mechanistic biology it is now necessary to distinguish carefully between effects mediated by the mother plant and thus strictly maternal, from those which are simply direct effects of environmental variation on the developing zygote itself. In practise the two can be differentiated through careful physiological and mechanistic studies coupled with clear genetic analysis demonstrating whether the maternal genotype, or that of the zygote, is key to response to an environmental signal. So here I will define 'Maternal Environment (ME)' strictly as *environmental effects involving direct perception by the tissues of the mother plant*, and mediated by either the genome or epigenome of the mother. Typically this involves modification of fruit and/or seed coat tissues, the production of signals from the mother to the maturing zygote, or inheritance of maternal epigenetic states through meiosis into the embryo or endosperm. I will distinguish this from effects of the 'Zygotic Environment (ZE)' which I will define as *environmental effects involving direct perception of environmental signals by tissues of the zygote* during seed development and maturation, and genetically under the influence of the genome or epigenome of the embryo and/or endosperm. Carefully distinguishing between ME and ZE effects will be necessary to breed for improved seed characters, especially in crops where F_1 hybrid seeds dominate the market.

Evidence for maternal influences on progeny seed germination

It has long been known that variation in the temperature during seed set strongly affects seed dormancy, with lower temperatures almost always resulting in lower germination. However, what is less clear is whether this really is a maternal process, or whether paternal alleles in the zygote also contribute. One important set of experiments that reveals the presence of signal transduction pathways in the mother plant is the use of environmental manipulations

before first anthesis to manipulate progeny seed germination. For instance, temperature during the vegetative phase can been shown to affect progeny seed dormancy in tobacco, oats and Arabidopsis (Thomas and Raper, 1975; Sawhney et al., 1985; Chen et al., 2014), the wide conservation of this affect across diverse angiosperm families showing that this is a very general phenomenon. Because there are no gametes in the vegetative phase these experiments show clearly that it is not just the ZE that affects seed dormancy, but that the temperature experience of one or more of the parent plants can be remembered across time and affect progeny seed properties. In addition to temperature variation, it has been shown that altitude and time of year can affect seed dormancy, probably through differences in ME and ZE during seed production (Fenner, 1991).

The role of the seed coat in the environmental regulation of dormancy

In seeds with physical dormancy such as many legumes, the seed coat acts to prevent water uptake by the zygotic tissues (Bolingue et al. 2010). Seed dormancy in legumes depends on pigments such as tannins, and light-coloured seeds are less dormant. In legumes therefore, seed size and seed dormancy are in general maternally inherited (Davies, 1975). In other species, most notably barley, the seed coat has been shown to be essential for creating a low oxygen environment in the seed, and removing the seed coat permits growth of the embryo at low oxygen concentrations (Lenoir et al., 1986). The key evidence that the seed coat is critical to the imposition of physiological coat imposed dormancy in Arabidopsis comes from the very low dormancy of *transparent testa* (*tt*) mutants (Debeaujon et al., 2000). Even when *tt* mutant seed is set at low temperatures, some alleles retain close to 100% germination (MacGregor et al., 2015), far surpassing the consequences of deleting *DELAY OF GERMMINATION1* (*DOG1*; Kendall et al., 2011), which can enter dormancy under the same conditions. However, when set at 16℃ there is no strict correlation between seed coat colour and dormancy, with some yellow seeded mutants showing much weaker phenotypes than others (MacGregor et al., 2015), and this raises the possibility that the tannins themselves are not the only seed coat factors affecting dormancy. Further evidence for this comes from *Spergularia diandra*, where yellow seeded varieties are more dormant than dark seeded varieties (Gutterman, 1994), although in this case the dark-seed forms appear to have some defects in integument development. In addition to colour mutants, other genetic lesions that affect the ovule integument or seed coat development also affect seed dormancy (Leon-Kloosterziel et al., 1994; MacGregor et al., 2015).

The second candidate for the imposer of coat-imposed dormancy is of course the endosperm. In a key paper Bethke et al (2007) showed that mechanically removing the seed coat from

dormant Arabidopsis seeds did not break dormancy, whereas breaching the endosperm did. This appears instead to favour the endosperm as critical to seed dormancy imposition. The important role of the endosperm has further support, including genetic evidence that reactive oxygen production in the endosperm promotes dormancy (Penfield et al., 2006), and more comprehensively that the endosperm synthesises and secretes ABA in dormant Arabidopsis seeds (Lee et al., 2010; Kang et al., 2015). It is possible that the seed coat is required not during imbibition, but during seed maturation to impose physiological dormancy, for instance by secretion of growth regulators, or by provision of materials to the cell wall of the endosperm which itself has been implicated directly in dormancy imposition (De Giorgi et al., 2015).

The seed coat is a highly plastic plant organ that responds to environmental signals. Depending on the species this response can lead to a change in the development or metabolism in the seed coat producing a change in dormancy observable upon imbibition under favourable conditions. The genus *Chenopodium* exhibits physiological seed dormancy that depends on seed coat thickness, which in turn is affected by environmental signals experienced by the mother plant during seed maturation. In *Chenopodium polyspermum* and *Chenopodium album* seed coat thickness depends on day length with shorter days leading to thinner seed coats and seeds with higher levels of germination at harvest (Karssen, 1970; Pourrat and Jacques, 1975). A similar effect on seed coat development in *Chenopodium bonus-henricus,* but this time altitude rather than photoperiod was the driver of seed coat development, with higher altitudes leading to thicker seed coats and lower germination (Dorne, 1981), and with high altitude seeds accumulating more polyphenols. These ME effects are often associated with effects on seed coat colour which have been observed in several species including legumes where the seed coat also imposes physical dormancy (Gutterman and Evenari, 1972). Other environmental signals that affect seed coat development include salinity and nutrient levels (Wang et al., 2012). Strikingly, Wang et al (2012) showed that the ratio of brown to black seeds produced by *Suaeda aralocaspicavia* plants depended not only on the maternal environment but also the colour of the seed coat of the starting seed. This shows that maternal effects can persist across whole generations in some cases. With a keen eye differences in coat colour between seed lots matured at different temperatures can be observed in the model species Arabidopsis, and measured with a multispectral imager (Figure 1). These are accompanied by changes in seed coat tannin levels, but no obvious effects on seed coat thickness (MacGregor et al., 2015). Measuring the precise impact of these changes in tannin levels on seed dormancy is a challenge because although it is clear the tannin is required for dormancy, separating the contribution of the impacts on tannin levels from impacts on other processes, such as effects on hormone levels and *DOG1* expression, is not straightforward. However, it is clear that Arabidopsis is a good model system for investigating

environmental control of seed coat development, although the importance of the process is likely to vary from species to species. In Arabidopsis as in other species changes in seed coat thickness or tannin content caused by temperature effects are associated with altered seed coat permeability to water and dyes (Gutterman, 1978; MacGregor et al., 2015),

The fact that a memory of past temperature can be used by plants to control seed coat properties, and that in some species the process is affected by photoperiod, are important clues that seed coat metabolism is influenced by genes involved in flowering time control. *FLOWERING LOCUS C* (*FLC*), circadian clock genes and *FLOWERING LOCUS T* (*FT*) control flowering in response to vernalisation and day length in Arabidopsis, and all have been shown to affect seed dormancy (Chiang et al., 2009; Penfield and Hall, 2009; Chen et al., 2014). *FT* expression in the phloem of siliques is more than 100-fold higher than in leaves (Adrian et al., 2010; Chen et al., 2014) and responds to ME signals that affect seed dormancy (Chen et al., 2014). In contrast, the Arabidopsis *FT* promoter is not expressed directly in seed tissues. Phloem-expressed FT-GFP fusions accumulate in the chalazal pole of the seed coat, indicating that the FT protein may be translocated to the seed to control dormancy, although transcript analyses show that FT also acts locally in silique tissues. The effects of *ft* mutation on the silique transcriptome can be phenocopied by exposing the vegetative tissues of the mother plant to lower temperatures (Chen et al., 2014). Together with genetic evidence that FT is necessary and sufficient for ME-regulated changes in seed coats and seed dormancy, this data show the central role of fruit-expressed FT in dormancy regulation by the ME. In *Capsella bursa-pastoris* flowering time was also linked to seed coat properties, with early flowering lines showing lower dormancy and having less seed coat mucilage (Toorop et al., 2012), and in *Ononis sicula* (Fabaceae) lengthening the photoperiod results in yellower seeds which in this case have more dormancy (Gutterman and Evenari, 1972). In Arabidopsis, flowering time genes can be directly linked to seed coat metabolism, with *ft-1* mutants showing increased tannin accumulation and increased expression of phenylpropanoid pathway enzymes necessary for tannin biosynthesis (Chen et al., 2014). These changes are associated with an effect on the transcript levels of major regulators of tannin biosynthesis in ovule integuments including *TRANSPARENT TESTA2* and the MADS box transcription factors *TRANSPARENT TESTA16* and *SHATTERPROOF2* (Figure 2).

In Arabidopsis studies disagree on whether photoperiod has an effect on dormancy, but our experience is similar to that of Donohue et al., (2005) in that standard photoperiod treatments used in plant physiology applied to the mother plant do not affect seed dormancy, and also do not affect *FT* expression (Adrian et al., 2010; Chen et al., 2014). This observation effectively rules of a role for FT synthesised in leaves in dormancy control. Yet the key photoperiod sensor protein *CONSTANS* is highly expressed in Arabidopsis siliques, so it is unclear why

FT expression in Arabidopsis fruits is unable to respond to photoperiod changes. A similar uncoupling of photoperiod responses was previously observed in cucumber where plants were daylength insensitive for flowering, but dormancy was promoted by long day signals applied to fruits (Gutterman, 1978).

Flowering time gene manipulation affects hormone metabolism in fruit and seeds

In addition to changes in the seed coat, flowering time genes also affect hormone metabolism in seeds and in this way could influence dormancy in some species. This could be particularly important in soft-fruited seeds such as tomato and cucumber where it has previously been shown that photoperiod affects accumulation of uncharacterised germination inhibitors in the pulp surrounding the seed (Gutterman, 1978a). The fact that photoperiod treatments can be applied to detached fruits and affect seed dormancy is good evidence that FT synthesised in fruits rather than leaves is important in dormancy control. ABA is a strong candidate for this fruit-derived germination inhibitor because in tobacco maternal ABA is important for seed dormancy regulation (Frey et al., 2004). This fruit-derived ABA can also be affected by nutrient status of the mother. Natural variation at *FLC* also influences ABA levels in seeds (Chiang et al., 2009), but this seemed to be directly in zygotic tissues because the effect was preserved in mature seeds. In addition, loss of FT increases the accumulation of both jasmonate and gibberellin (GA; Chen et al., 2014). Notably, *ft-1* mutant seeds accumulate higher levels of GA despite being more dormant than wild type, demonstrating that FT has the potential to increase dormancy, as well as decrease dormancy, acting through different mechanisms. It is also relevant that control of seed dormancy by oxylipins has been proposed to occur via an FT orthologue MOTHER OF FT AND TFL1 (MFT; Dave et al., 2016). This is an important observation because it is likely that different overlapping signalling cascades occur in different seed compartments using overlapping gene networks leading to different outcomes controlling dormancy. In embryos this is likely affects levels of hormones and control of hormone signal transduction. For instance, FLC has not only been linked to regulation of ABA metabolism through *CYTOCHROMEP450 707A* (*CYP707A*) genes (Chiang et al., 2009; Deng et al., 2011), but also to indirect regulation of *GA3OX1* via both *TEMPRANILLO1* and *APETALA1* (Mateos et al., 2015). It is very likely that understanding the environmental control of seed germination will require understanding in detail links between flowering time pathways which carry environmental signals and hormone metabolism and signalling. Interestingly, maternal control of ABA levels has also been linked to the regulation of of seed coat pigmentation (Frey et al.,2004; Gu et al., 2011) and Arabidopsis *della* mutants have reduced seed coat

permeability (Chen et al., 2014), showing that hormone production and signalling also play a role in the development of the seed coat during seed maturation.

Seed dormancy and germination control by the Zygotic Environment

The clearest direct effect of the ZE on seed dormancy is the manipulation of light quality during seed maturation. Changing the red/far-red ratio during seed production changes the dormancy of seeds produced on the mother plant (Hayes and Klein, 1974; Cresswell and Grime, 1981). Importantly, giving far-red during seed maturation induced a red light requirement for germination, and this depended on the green colour of the tissues surrounding the zygote (Cresswell and Grime, 1981). Thus suggests that the perceptive tissue is the zygote within the green fruit tissues. It is hypothesised that in the embryo or endosperm the Pr:Pfr ratio of phytochrome states at maturity can be affected by light treatments given during seed maturation, and that this can subsequently affect light requirements upon imbibition. However, phytochromes do strongly affect *FT* expression in leaves (Halliday et al., 2003), so it is also possible that phytochrome has an overlapping dormancy-imposing role during seed development. In Arabidopsis there are five phytochromes (phyA-E), and loss of phyA, phyB and phyE causes a strong increased dormancy phenotype or a poor germination response to far-red light (Hennig et al., 2002). However, if seeds are matured at cooler temperatures then phyD becomes important for the response to dormancy-breaking temperature treatments (Donohue et al., 2008). This increase in importance for phyD may be explained by the fact that low temperatures during seed maturation strongly reduce *phyB* and *phyE* gene expression in seeds (Kendall et al., 2011). This underlines the close integration of light and temperature signalling pathways in Arabidopsis (Halliday et al., 2003; Penfield et al., 2005; Koini et al., 2009). As well as light quality, light intensity during seed maturation has been shown to affect seed dormancy in Arabidopsis (He et al., 2016).

Measuring the composition of developing and mature seeds, especially in species in which maternal tissues undergo apoptosis during the final stages of desiccation, is an excellent way to understand effects of ZE on seeds (Kendall et al., 2011; He et al., 2014; Righetti et al. 2015; He et al., 2016). Temperature during seed maturation has a strong effect on *DOG1* transcript levels in mature seeds, and also effects *CYP707A2* gene expression, GA and ABA levels (Kendall et al., 2011; Chiang et al., 2011). Because genetics shows that DOG1 and ABA affect seed dormancy from the zygotic genotype (Koornneef et al.,1982; Alonso-Blanco et al., 2003) it is possible that these are direct impacts from temperature perception in the zygote itself. Measurement of the effects of light intensity, temperature and nitrate on metabolites in mature seeds show that these signals directly affect the metabolome of the zygote (He et al., 2016), suggesting they act in zygotic tissues. By combining an analysis of several Arabidopsis lines

with different levels of dormancy He et al (2016) showed that these effects on metabolites are dependent on the genotype, and that genotypes with similar dormancy levels had similar metabolomes. This suggests that metabolic composition is a consequence of dormancy state or the action of gene networks controlling dormancy state, rather than a causative factor of differences in dormancy levels. Thus we cannot rule out that some of the environmental signals controlling seed composition, dormancy and longevity in response to nutrient levels or light intensity also emanate from the mother plant.

Aside from DOG1, the best candidate for a central regulator of temperature response to the ZE is *MOTHER OF FT AND TFL1* (*MFT*; Nakamura et al., 2011). In wheat *MFT* is expressed in the embryonic tissues and is strongly up-regulated by low temperatures during seed maturation (Nakamura et al., 2011). Loss of MFT leads to lower dormancy indicating that MFT is a germination inhibitor, and variation at MFT underlies variation in dormancy among East Asian cultivars of wheat (Chono et al., 2015). Taken together the data show that MFT is a low temperature-induced inhibitor of seed germination. In Arabidopsis the role of MFT as a germination inhibitor is conserved and *MFT* is a direct target of the SPATULA bHLH transcription factor, which in turn is necessary for normal temperature responses in Arabidopsis seeds (Penfield et al., 2005; Vaistij et al., 2012). In Arabidopsis *MFT* is only weakly temperature-regulated at the transcript level compared to wheat (Kendall et al., 2011). Interestingly, SPT is also a regulator of fruit development (Heisler et al., 2001), further underlining the evolutionary links between environmental signalling pathways in the fruit, integuments and zygote. *MFT* expression is strongly up-regulated by the germination inhibiting hormone 12-oxo-phytodienoic acid (OPDA; Dave et al., 2016) suggesting the involvement of oxylipin hormones in temperature responses in seeds, as is the case during cold acclimation (Hu et al., 2013). MFT acts to control seed dormancy in a complex gene network including ABI5, DELLAs and hormone metabolism (Dave et al., 2016; Figure 2) that still requires more detailed analysis, and this analysis probably needs to extend beyond Arabidopsis where temperature effects on *MFT* expression are apparently attenuated. MFT is highly conserved among major crops suggesting it is a good target for dormancy manipulation in crop species.

Conclusions

It is interesting that the major effects of the ZE on seed dormancy involve temperature effects on *DOG1*, *MFT* and phytochrome because these appear to mimic those seen in buried seeds during seed dormancy cycling, the induction and loss of secondary dormancy (Footitt et al., 2011). So it is possible that these ZE effects result from a partial activation of secondary dormancy-regulating processes during seed maturation in the zygote. These processes work alongside truly maternal pathways in determining the final dormancy status of the mature

seed. These maternal processes clearly involve modification of fruit and seed coat tissues, but there are also maternally-derived mobile signals such as production of hormones and FT protein that have the potential to interact directly with zygotic tissues to control seed properties. Finally it is clear that some epigenetic states are inherited from mother to zygote, including imprinted genes and genes such as *FLC* which is strongly expressed and temperature sensitive in all tissues of the seed (Sheldon et al., 2008). Because there are high levels of cross talk between seed tissues it is very likely that maternal and zygotic environmental signal transduction pathways interact at several levels that remain to be discovered.

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Figures

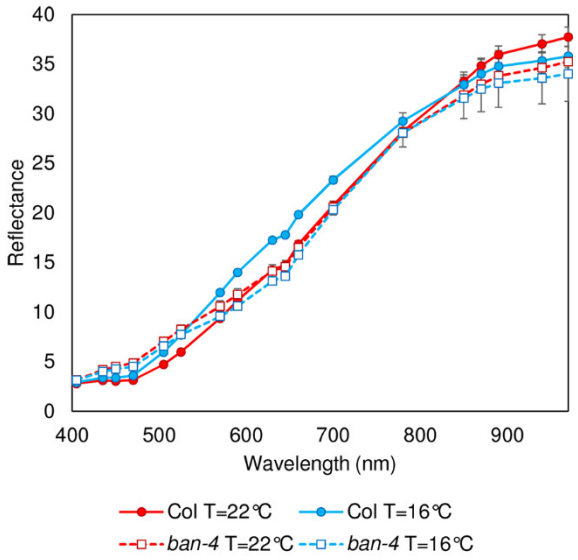

Figure 1. Replicate seed batches of Arabidopsis thaliana Col-0 accession produced at either 16℃ or 22℃, to show seed coat colour differences observable between the two treatments. Shown is the multispectral quantification of light reflectance from Arabidopsis Col-0 and the tannin-deficient *banyuls-4* (*ban4*) mutant. Differences in reflectance seen in wild type seeds set at different temperatures are no longer observed in *ban-4*.

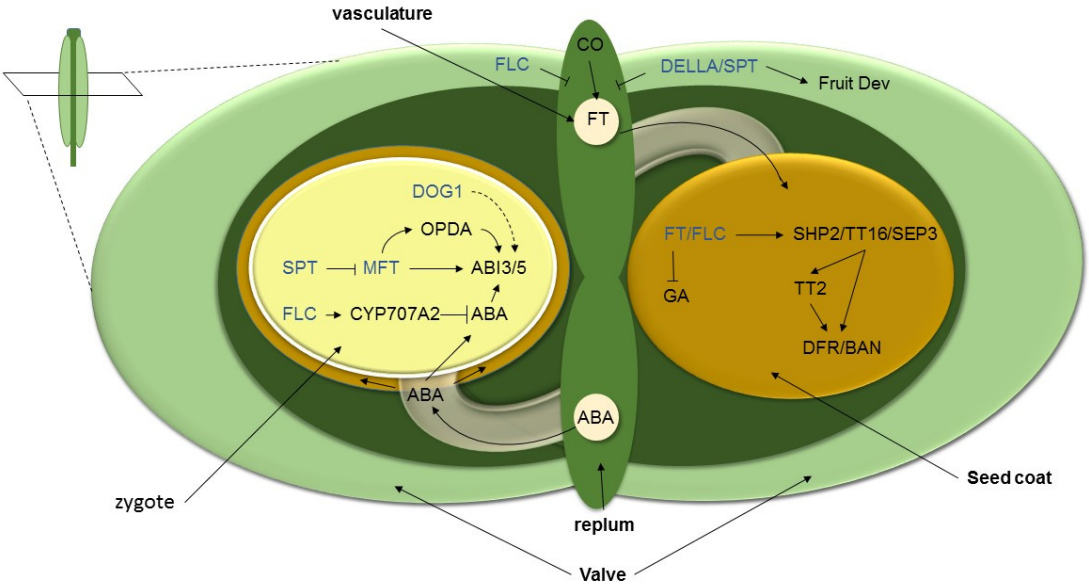

Figure 2. Summary of characterised processes mediating control of seed dormancy and germination by environmental signals during seed production. Diagram shows cross section of an Arabidopsis fruit revealing the internal structure including seeds. On the left processes which control responses to the ZE, the seed on the right processes controlling seed germination from the ME. Supporting data is to be found in Chiang et al., 2009; Penfield and Hall, 2009; Chen et al., 2014; Kendall et al., 2011; Chiang et al., 2011; Deng et al., 2011; Mateos et al., 2015; Kaufmann et al., 2009; Vaistij et al 2012; Nakamura et al., 2011; Dave et al 2016; He et al 2016). Temperature-regulated genes are shown in blue.